THE CLINICAL IMPORTANCE OF ASEPTIC INFARCTION OF THE KIDNEY.

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Infarction of the kidney is a condition more familiar to the pathologist than to the clinician, as the paucity of clinical literature on the subject attests. That knowledge of its manifestations concerns the surgeon was first impressed upon me when I saw two patients suffering with the condition within a period of two months. Both were admitted to the surgical service as cases in urgent need of operation. Schmidt, in his admirable report of three cases from Neusser's clinic, contrasts the frequency of renal infarcts found at autopsy with the rarity of their clinical recognition. He says that the diagnosis is important for differentiation from conditions requiring early surgical intervention.

The following histories of the cases which were encountered illustrate the practical importance of considering the diagnosis of renal infarction in patients with valvular disease who present the symptoms of an acute intra-abdominal disorder.

Case I.—M. Z., male, aged thirty-one years, was admitted to the surgical service on the evening of February 16, 1921, with the following history: Two weeks previous to admission he was suddenly seized with sharp pain in the right lower quadrant of the abdomen. The pain lasted about three hours and disappeared spontaneously. It did not radiate nor was it accompanied by vomiting. Two days later he was seized again with severe abdominal pain, cramplike in character, associated with nausea and vomiting. During the following week he had discomfort in the right lower quadrant of the abdomen, with some temperature, and was confined to bed. Three days ago he left his bed, was seized with violent pain in the left flank and vomited. The pain has been present in this location since the onset. It has been constant in character but intensified by bodily motion and deep breathing. The pain on the day of admission radiated downward to the left testicle. For the past week he has had frequency of urination, voiding about every hour. No dysuria, hematuria, nor pyuria.

Examination soon after admission showed a well-developed, well-nourished man, acutely ill, and apparently in severe pain. Pupils and reflexes were normal, the tongue dry and coated, the breath foul. There was distinct voluntary limitation of respiratory excursions. There were dulness and diminished breathing at the

left base posteriorly, but the lower border of the lung came down with deep inspiration. The heart was somewhat enlarged, the apex-beat was in the fifth space 12 cm. from the midsternal line, where a distinct presystolic thrill was felt. A well-marked rumbling presystolic murmur was heard at the apex and transmitted for a short distance into the axilla. The second pulmonic sound was accentuated. The pulse was 90, regular and of good quality. The abdomen was rigid, specially on the left side. There was tenderness over the left lower ribs in the axilla and in the left hypochondrium below the costal border. There was tenderness in the left costovertebral angle also. The left lumbar muscles were rigid. Rectal examination was negative. Temperature, 101.8°F. Blood count: Leukocytes, 20,000; polymorphonuclears, 82 per cent. Urine on admission: Clear; specific gravity, 1020; albumin present; no sugar. Microscopic examination: A few hyaline casts and occasional leukocytes; no erythrocytes.

The differential diagnosis lay between perinephric abscess, subphrenic abscess, infarction of the spleen and infarction of the left kidney. Because of the repeated attacks of pain, first on the right and then on the left side, and the presence of a definite mitral stenosis and of albuminuria, a diagnosis of renal infarction was made. It was therefore decided not to operate immediately.

Under observation the anterior tenderness and muscular rigidity subsided almost completely. The costovertebral tenderness and lumbar rigidity became predominant. The temperature on the day after admission rose to 103°F. Roentgen-ray examination showed no calculus in any part of the urinary tract. Cystoscopy on February 19 showed no abnormality of bladder or ureteral orifices and no obstruction to ureteral catheters. The indigo-carmine appeared in good concentration in twenty minutes on the left side and in somewhat less concentration on the right side in about the same time. Specimens collected for bacteriology proved sterile. Both specimens showed many granular casts and scattered red blood cells (possibly traumatic in origin). The Wassermann test was negative and the blood culture taken on February 17 showed no growth.

February 19, it was decided to explore the left kidney because of the temperature (102°F.) and the persistent and localized tenderness in the left costovertebral angle. The operation performed by the house surgeon, Dr. Jones, revealed a somewhat enlarged kidney with no gross perinephric changes. The kidney was delivered into the wound easily. It presented at its middle and near the lower pole two grayish-white, pearly, circumscribed areas, about 3 cm. in diameter, firm in consistency with surface slightly above the level of the surrounding parenchyma and a narrow injected zone about each of them. Specimens were removed from these areas for pathologic examination. The kidney was

decapsulated and the wound closed with a small rubber-dam drain behind the kidney.

On the day following the operation the temperature rose to 103.2°F. and then gradually returned to normal by lysis, reaching 99°F. by the rectum on the sixth day. All pain and tenderness disappeared. The urine on the day following the operation showed gross blood, and on the sixteenth day after operation still showed a few scattered erythrocytes. The phthalein test on February 24 showed 15 per cent excretion in two hours, and the blood chemistry on February 22 showed urea nitrogen 37.8, incoagulable nitrogen 77, uric acid 2.6, creatinin 1.4. The tisssue removed at operation proved to be part of an anemic infarct of the kidney. No microörganisms were found in the stained sections.

The wound healed kindly and the patient was discharged free of all symptoms on March 7, 1921, and has remained well up to the present time. Diagnosis on discharge was mitral stenosis and anemic infarcts of the left kidney.

Case II.—J. K., female, married, aged forty-four years, was admitted to the surgical service on the afternoon of April 6, 1921, with the following history: About two hours after eating a light lunch on the day before admission she was suddenly seized with severe abdominal cramps. Toward evening the pain localized in the right upper quadrant of the abdomen, where it became constant and intense, occasionally radiating to the back and shoulders. One-half hour after the onset she vomited. The pain was increased by bodily motion, straining at stool and deep breathing. The bowels moved twice with saline catharsis. The patient had been feverish since the onset. There was no history of previous similar attacks and no urinary symptoms. The patient stated that when nine years old she had chorea which was followed by heart trouble. Since that time she had suffered with palpitation and dyspnea on moderate exertion. About one year ago she had an attack of unconsciousness and aphasia lasting several days, but this cleared up completely. She had several abdominal operations for gynecologic conditions.

Examination showed an obese female acutely ill, cyanosed and dyspneic. She was apparently in severe pain. The pulse was rapid, 120 to 130, and very irregular. The temperature was 102.2°F. The abdomen was moderately distended, but soft, except in the right upper quadrant, where there was a moderate amount of rigidity of the rectus. Marked tenderness was elicited in the right hypochondrium and in the midepigastrium, and slight tenderness in the right costovertebral angle. There was hyperalgesia of the skin in the distribution of the 11th, 12th thoracic and 1st lumbar nerves of the right side. The right lumbar muscles were

rigid. The lungs showed no detectable abnormality. The heart was enlarged toward the axilla, the apex-beat being in the 6th space in the anterior axillary line. Cardiac action was absolutely irregular, but no murmurs could be detected. The pulmonic second sound was accentuated. Blood examination: Leukocytes, 24,000; polymorphonuclears, 91 per cent. Urine: Clear; specific gravity, 1018; large amount of albumin; no sugar. Microscopic examination showed many erythrocytes and but few hyaline and granular casts.

The differential diagnosis lay between acute cholecystitis with possible pancreatitis, acute appendicitis, intestinal obstruction due to postoperative adhesions and infarction of the right kidney. The presence of a cardiac lesion and the urinary findings strongly pointed to the last diagnosis. It was therefore decided to postpone surgical interference.

On the following day the tenderness in the right hypochondrium almost completely disappeared, while that in the costovertebral angle became exquisite. Both conjunctivæ showed a few small recent petechiæ, one in the left conjunctiva showing a prominent pearly central spot. On April 8 all anterior tenderness had disappeared, the posterior tenderness persisting. The phthalein test on April 10 showed 35 per cent excretion in two hours. Blood chemistry on April 7 showed: Urea nitrogen, 33.6; incoagulable nitrogen, 59.5; uric acid, 3.8; creatinin, 1.9; cholesterin, 0.18 per cent. Blood Wassermann was negative. Blood culture taken on April 6 was negative.

With the subsidence of acute pain the temperature gradually fell by lysis, reaching 99.2°F. on the seventh day after admission. The urine continued to show large amounts of albumin, casts and a few erythrocytes until April 18, 1921, when merely a trace of albumin was present and a few leukocytes formed the only abnormal elements. On April 12 the patient was transferred to the medical service of Dr. Libman, who concurred in the diagnosis. On April 13 the blood-pressure was systolic 160 and diastolic 80. The leukocytes were 10,200 with 74 per cent polymorphonuclears. Cystoscopy on April 14 showed no abnormality of the bladder or the ureteral orifices. Good indigo-carmine excretion appeared on both sides in eighteen minutes. Specimens collected for bacteriology showed no growth. During the period of observation of seven weeks on the medical service there was no rise in temperature and no further embolic phenomena. The cardiac irregularity persisted in spite of therapy. The electocardiogram confirmed the clinical finding of auricular fibrillation. The patient was discharged in fair general health with no urinary abnormality except for a faint trace of albumin. Diagnosis: Mitral stenosis and insufficiency, auricular fibrillation and infarction of the right kidney.

It will be noted that these cases have certain features in common. Both patients had mitral valvular disease. The onset in each case was sudden, with abdominal cramps, nausea and vomiting. The pain, then localized to the hypochondriac region, became constant, but was intensified by bodily motion and deep breathing. Tenderness and muscular rigidity predominated anteriorly at first, and later, as the attack subsided, became localized to the lumbar region posteriorly. Albuminuria, cylindruria and erythrocyturia (in one) were present without signs of cardiac decompensation nor the usual manifestations of nephritis. In the absence of pyuria the fever and the marked polymorphonulcear leukocytosis added to the difficulties of diagnosis.

Welch, referring to the question of fever in aseptic embolism, says, "I am not aware of any conclusive observations which show that fever may be produced in this way in human beings." He includes fever, nevertheless, among the general symptoms of renal infarction. Robert Sprinz, in his Inaugural Dissertation at Wurzburg, in 1885, concludes that embolism can produce fever regardless of the nature of the embolized material, and that the fever is due to the embolism, not to the absorption of necrotic tissue from the infarct. Bock has shown experimentally that fever can be produced by the injection into the blood stream of sterile, chemically indifferent particles. Billroth made similar experiments in connection with his studies on wound fever.

Slight fever is noted by Schmidt and Halperin and more marked febrile movement by Riebold. Both Halperin and Riebold report a leukocytosis of over 20,000. The cases here reported show definite febrile movement and well-marked leukocytosis, with all evidence pointing to a bland aseptic infarction.

Senator states that urinary changes occur only with very large or numerous small infarcts and that the presence of blood is essential. Welch, however, says the amount of blood is usually only moderate or even microscopic, and may be absent; hematuria being less common than albuminuria. Chedevergne emphasizes the latter statement in his Paris thèsis, 1901, "De l'albuminurie symptomatique de l'infarction rénal."

Schmidt finds albuminuria constant though transient and erythrocyturia uncommon. In complete unilateral infarction the urine may be altogether negative. The absence of formed elements in some cases may be explained by their retention in the infarcted areas. He emphasizes intense oliguria or anuria in bilateral infarction.

The following abstracts are those of cases reported in the literature, grouped according to etiology.

A. Cases of thrombosis of the renal arteries.

1. Due to trauma.

Von Recklinghausen, 1861.

Boy, aged eight years, died eight days after a fall. Autopsy revealed severe internal injuries. Circular injury to the coat of the left renal artery two lines in length and one in width; ½ cm. below this was a large red thrombus; all branches were thrombosed. The kidney was entirely necrotic.

2. Due to infectious diseases (acute arteritis?).

Juhel Renoy.

A girl, aged sixteen years, recovering from scarlatina. Pain in both lumbar regions on the second day of normal temperature, not severe. Complete anuria for six days; death was preceded by several convulsions.

Autopsy. No cardiac or aortic lesions. Both kidneys were entirely necrosed. Multiple thrombi in the renal arteries and glomerular tufts. Bacteriologic examination negative.

3. Due to subacute and chronic diseases (arteritis, periarteritis).

Halperin, 1908.

In 1905, a man aged forty-two years, was admitted to Micheal Reese Hospital, Chicago. One year ago he had pain in the left big toe after exposure to cold. Five weeks ago the pain recurred and the foot and the lower leg became livid. Gangrene set in and progressed upward. The heart was negative and the urine was normal. Three weeks after admission, fever, sweats, rigors and cough appeared with ascending thrombo-phlebitis of the leg and areas of dulness in the lungs. Urine now showed albumin and casts but no blood. Amputation resulted in recovery.

July 2, 1907. Two weeks' history of pain in the left upper quadrant, vomiting and pain in the left calf. While in the hospital he had two severe attacks of lumbar pain, accompanied by vomiting, headache, fever and leukocytosis of 28,000. Urine: Marked trace of albumin; hyaline, granular and cellular casts, few red blood cells and some hemoglobin. Tenderness in both flanks.

October 6. Severe stabbing pain in the left lumbar region radiating forward to the median line, profuse vomiting and severe headache. The left kidney region was tender. The peripheral vessels were sclerosed. The pain gradually subsided but the urine was suppressed.

October 7. One dram of urine showed much albumin, many pus cells, but no casts or red cells.

October 8. Blood-pressure, 210; temperature, 101°F. Signs of cardiac insufficiency.

October 9. Nine drams of urine obtained showed pus, albumin and hemoglobin. Leukocytes, 16,500; temperature, 99°F. Left kidney explored by Dr. John B. Murphy and found dry, pale gray, practically bloodless.

October 11. Died after six days of almost complete anuria.

Autopsy. Left kidney of normal size showed many cicatricial depressions. In midzone a whitish gray infarct, 7½ cm. in length extending from the cortex to the pelvis and occupying two-thirds of the kidney. The renal artery wall was thickened with a red thrombus at the bifurcation. The right kidney was very small and irregular. There was a large gray infarct of the upper third. There were small cortical infarcts. The renal artery was more thickened than the left, with definite contraction (1 mm. lumen) vessels and branches plugged with adherent red and white thrombi.

Comment. Nationality of patient not given. Microscopic studies of vessels not reported. History suggests thrombo-angiitis obliterans.

Pic and Bonnamour, 1913.

Female, aged sixty-nine years, was admitted February 17 to the Hotel Dieu, Paris, for paralysis of the left face and arm. She presented signs of aortic insufficiency, marked arrhythmia and atherosclerosis. The urine was small in amount and decreasing; there was much albumin.

February 24. Temperature rose to 39° C., when sudden pain in the left flank and abdomen set in.

February 28. Exitus after progressive alleviation of pain.

Autopsy. Dilated atheromatous aorta. Left renal artery and branches completely occluded by a thrombus. Kidney completely infarcted. Pathologist attributed thrombosis to disease of the renal artery and not to embolism.

Manges and Bæhr, 1921.

Man, aged thirty-nine years, was admitted to the surgical service of Dr. Beer, Mt. Sinai Hospital, New York City, June 6, 1919, apparently suffering from some acute abdominal condition. There was a history of cramp-like pains in the calf of both legs radiating to the feet. As these subsided he experienced severe pain in the right lumbar region radiating into the testis, and a few days later had a similar pain on the left side accompanied by hematuria. Six days before admission he had severe epigastric pain, abdominal distention and fever of 102°F. Cystoscopy and the roentgen-ray were negative. Leukocytes, 36,000; polynuclears, 75 per cent. After four days the temperature dropped to normal but leukocytosis persisted. Blood-pressure, 160/90. Blood chemistry: Urea nitrogen, 35; incoagulable nitrogen, 95. Urine negative except for a few red blood cells. P. S. P., 64 per cent.

June 19. Exploration by Dr. Buerger for suspected retroperitoneal abscess revealed periarteritic nodules on the mesenteric vessels. He was then transferred to the medical service, where he developed nodules on the peripheral vessels. In the course of four months he developed signs of progressive renal insufficiency and died of a terminal bronchopneumonia.

Autopsy showed extensive periarteritis nodosa involving many of the small and medium-sized arteries. The most extensive lesions were found in the kidneys. The right kidney showed dense perinephritis, a false aneurysm in the cortex, occlusion of nearly all the renal arterial branches by thrombi. The left kidney was small, its surface studded with large depressed scars the result of numerous old infarctions, and its vessels extensively diseased. The cortical markings were completely obscured in both organs.

B. Cases of embolism of the renal arteries:

Traube, 1853.

Machinist, aged eighteen years, with aortic insufficiency.

October 14. At 1 A.M., sudden onset with pain, awoke from sleep. Pain intensified by pressure over the right kidney, by bodily motions and cough; lessened by lying on the right side and by leeching. It radiated into the right thigh. The urine before the attack was small in amount dark and sedimented. After the attack it was diminished in amount and was dark, with uric acid sediment but no albuminuria. Pressure sensation in the bladder region, dysuria, increased pulse-rate and restlessness.

October 19. Vomiting and collapse, fear and restlessness and air hunger. Died nine days after onset.

Autopsy. The aortic valves were incompetent, with warty vegetations. Splenic infarct the size of a hazelnut. Old small infarcts of both kidneys, but the right showed a large infarct extending from the convex border to the pelvis, two inches long and prominent.

Bartels, 1870.

Boy, aged eight years, previously tracheotomized for croup, suddenly developed pain in the region of the spleen, which became enlarged. Several days later pain in the left thigh, followed by paresis of the extremity and death in four days. There were no urinary symptoms.

Autopsy. Large splenic infarct. Large left renal infarct; renal capsule and perirenal fat thick and swollen. The renal artery and its branches were thrombosed. Healing lesions of diphtheria. Thrombus in the apex of the left ventricle, with ragged, broken surface (probable source of emboli).

Von Leube, 1895.

Cabinet-maker, aged seventeen years. Acute endocarditis (aortic insufficiency) in the course of acute articular rheumatism. The patient's urine contained albumin and blood. The left kidney region was very tender. No edema. Blood disappeared in two days and the albumin in six days. On March 6, pressure in the chest, headache, and fever of 38.6° C. appeared. The urine again showed

albumin and blood and the left kidney again became tender. Urinary changes persisted seven days, then all the symptoms subsided. Diagnosis of renal infarction was made. The patient recovered.

Chedevergne, 1902.

Male, aged twenty-two years, with aortic and mitral disease. Urine negative over long periods of observation in two hospitals. Returned six days after discharge with marked dyspnea and arrhythmia but without passive congestion. Urine now showed a large amount of albumin without casts. Sudden pain in the popliteal space of the right leg. Urine decreased in amount without increased concentration; the albuminuria increased. Gangrene of the right leg at first dry, then moist supervened, and death ensued with septic phenomena.

Autopsy. Mitral stenosis and secondary insufficiency of the aortic and tricuspid. Kidneys showed several deep depressions. The left kidney showed a recent infarct the size of an olive. The right femoral artery was occluded.

Schmidt, 1901.

Laborer, aged forty years; had rheumatism, pleuritis and carditis four years ago.

September 25. The patient was admitted with dyspnea, cyanosis and venous engorgement of the lungs and the liver.

October 10. Sudden pain in the right flank, increasingly intense.

October 11. Tender in the right lumbar region posteriorly and under the liver anteriorly; vomiting, hiccough, constipation.

October 13. Pain was aggravated by motion, breathing, vomiting, etc., and was relieved by lying on the affected side.

October 14. Pain disappeared but the lumbar tenderness persisted.

October 17. Peritoneal friction rub was felt anteriorly below the liver. The urine was not diminished; no changes. No fever. Sudden exitus the next day.

Autopsy. Healed endocarditis of the mitral valve. Extensive anemic infarct of the right kidney with hemorrhage into the renal capsule.

Schmidt.

Female, aged twenty-one years, had recurrent polyarthritis with cardiac involvement. Two years ago had hemiparesis, which gradually cleared up.

December 16, 1899. Admitted, complaining of anorexia, attacks of dizziness, headache and precordial pain. Findings, delicate individual, with slight flush and cyanosis. Mitral stenosis and insufficiency. Urine: 1022; amount, 1000 cc. Nucleo-albumin+ serum-albumin—No fever.

February 26, 1900. Discharged improved.

November 6, 1900. Readmitted, with slight edema of the legs.

December 2. Severe pain in the gall-bladder region, radiating to the lower thorax and flank; incessant vomiting and marked dyspnea; tenderness in the right flank.

December 3. Similar attack on the left side. Pain was relieved by lying on the belly, but was increased by deep breathing and movement of the right hip.

December 4. Almost complete suppression; hiccough; constipation; comfortable only with knees flexed.

December 6. Retention and oliguria; obstipation; headache; hiccough.

December 7 to 9. Rentention and oliguria persist. Gastrointestinal disturbance continued.

December 16. Progressive loss of weight and strength.

December 21. Exitus.

Highest temperature was 39° C. (December 4). There was one chill.

(December 5). Urine was entirely negative before the attacks. After the attacks there was marked oliguria and albuminuria, with red bloods cells. No casts or renal elements.

Autopsy. Healed mitral lesion with lime deposit. Numerous infarcts of the kidneys, specially the left. Numerous large infarcts of the spleen.

Schmidt.

Man, aged forty-five years, with mitral stenosis and insufficiency, who had responded to digitalis in several periods of decompensation.

December 6, 1901. Sudden attack of pain in the right upper abdomen with vomiting, dizziness and collapse. Pain localized to the right kidney region a few hours later.

December 7. Similar pain but less severe in the left kidney region. The pain was deep seated, constant and made worse by bodily motion and deep breathing. Tender to percussion over both kidneys and to pressure anteriorly below the costal border. Anorexia, vomiting and constipation, which persisted four days. Ileocecal region was tender. Great difficulty in voiding.

December 10. Leukocytes, 11,000. Temperature, 38.3°C.

December 16. Apparently well.

December 17. Epileptiform seizures followed by coma. Signs of right hemiplegia. Pulse in the left radial and brachial regions disappeared. Exitus.

Urinary changes were of interest. Oliguria with onset of renal infarction. Postembolic polyuria hitherto unobserved was noted. Only a few red cells were in the sediment. Definite albuminuria.

Epithelial casts of polygonal cells with red cells attached to each cast were found.

December 6, 7, 8. Casts of brownish color covered with urate, waxy-like casts and a few free red blood cells.

Autopsy. Old double mitral lesion, thrombus in the left auricle, multiple anemic infarcts of both kidneys. Infarcts of spleen. Embolus in the left Sylvian and left brachial arteries. Erosions of gastric mucosa.

Riebold, 1905.

Housemaid, aged twenty-nine years, suffering with cardiac decompensation and irregularity, the result of rheumatism seven years previously. Mitral stenosis and insufficiency. Improved under digitalis.

October 11. Sudden severe pain in the left flank, with nausea, vomiting and collapse. Pain constant, localized, aggravated by bodily motion and deep breathing. Abdomen distended, tender, specially in the left upper quadrant, with marked skin hyperalgesia. Marked constipation, no passage of gas. Temperature, 37.6° C.

October 12. Continued vomiting and pain. Spleen enlarged. Retention, but catheter yielded 500 cc of normal urine. Leukocytes, 21,600; temperature, 38.9° C.

October 13. Less pain, no vomiting, but obstipation with tenderness in the left upper quadrant.

October 14. Bowels moved and urine voided, but severe pain reappeared in the left flank and also appeared in the right flank.

October 16. Sudden right hemiparesis.

October 17. Exitus.

Autopsy. Left kidney; upper half completely infarcted, multiple small infarcts in the lower half. Perirenal tissue thick and edematous. Embolus lodged in the renal artery. Right kidney: Two cherry-sized fresh infarcts. Spleen: Wedge-shaped anemic infarct, the surface of which showed fibrinous exudate. Embolism of the basilar artery. Double mitral lesion.

Von Jagic, 1915.

Female, aged thirty-nine years, with postrheumatic cardiac disease; two weeks prior to admission, February 10, 1914, had an attack of pain in the left side of the abdomen, cramp-like and persistent. She presented signs of mitral and tricuspid disease, marked abdominal distention, with tenderness in the left lower quadrant, but no rigidity or rebound sign. Evening temperature, 39.2° C. Urine: 500 cc; specific gravity, 1023; much albumin, no blood. After three or four days there was a subsidence of pain and temperature.

February 14. Sudden rise in temperature to 38° C.

February 16. Repeated vomiting and severe pain on the right side, and a tender mass below the costal border.

February 27. Sudden exitus.

Autopsy. Mitral and tricuspid stenosis. Thrombi in the left auricle and ventricle. Emboli in both renal arteries, with anemic infarction of large areas of parenchyma.

C. Cases of doubtful etiology.

Gayet and Favre, 1914.

Female, aged thirty-four years, had typhoid with albuminuria in 1904. Vague renal pain simulating stone colic appeared in 1908. In 1913, urinary symptoms and attacks of colic led to cystoscopy. The right kidney function was half that of the left, some pus and colon bacilli. Roentgen ray was negative.

January, 1914. *Operation.* Right nephrectomy. Kidney showed infarct of half the kidney with small calcific lesion at the center. Was it due to typhoid or was it a sclerocyst about a small calculus?

Rathbun, 1916.

Russian teamster, aged thirty years, noted frequency and urgency of the urine one and a half years ago. One year ago had dull pain in the left flank with radiation along the ureter, growing progressively worse. The left kidney was palpable and tender to pressure and percussion. All examinations were negative. Equal and good excretion of P. S. P. from the kidneys. Congestion of the posterior urethra was relieved by treatment, but lumbar pain persisted.

May 5, 1916. *Operation.* Kidney enlarged and showed several hard lumps. Organ split and four lumps of fibroid consistency noted. Kidney had to be removed later for hemorrhage, and lesions were found to be infarcts with plugged vessels and coagulation necrosis. The patient had no cardiac lesion. No abnormality of urine was detected preoperatively.

Comment. Note strong resemblance to pseudogummatous lesions described by Beriel and Devic (les sequestres des reins).

Etiology. The immediate cause of renal infarction is the shutting off of the blood supply to smaller or larger areas of parenchyma. This may be due to thrombosis in the renal artery or its branches brought about by trauma, acute arteritis in the course of an infectious disease, chronic arteritis, atherosclerosis or periarteritis nodosa. Embolism is a more common cause of infarction. Chronic valvular disease was the cause of embolism in 10 cases, of which 2 were aortic and 8 mitral. The mitral lesions were stenotic and in 2 thrombi were found in the left auricle. The problem of embolic phenomena in valvular disease has been studied by Libman in connection with subacute bacterial endocarditis in the healing

or healed stage. In his opinion valvular defects *per se* do not provide emboli which produce clinically recognizable phenomena. When such phenomena do occur in patients with chronic valvular disease and bacteria-free blood the presumption is strong that the rheumatic infection has been followed by a bacterial invasion which has gone on to spontaneous healing.

It is important to remember that while infarcts occurring in the active bacterial stage of the disease do not suppurate, nevertheless the emboli producing these lesions are not aseptic.*

Aseptic emboli may come from thrombi in the pulmonary veins or the left auricle; from atheromatous lesions, ulcers, fibrinous deposits, thrombi or calcified vegetations on the mitral and aortic valves, or from atheromatous lesions of the aorta. Paradoxic emboli passing from some peripheral vein through an open foramen ovale or open septum may produce infarction.

Such conditions as aneurysm of the renal artery, thrombosis of the renal veins and infarction of the renal cortex in eclampsia do not come within the scope of this paper, as their clinical manifestations are different.

Onset. In the cases associated with chronic valvular disease the onset is sudden with severe pain in the abdomen and flank accompanied by vomiting, fever and more or less prostration and signs of collapse. In a few cases there was a history of mild indefinite attacks on the same or opposite side.

Pain. The first symptom is sudden, severe pain referred to either hypochondrium below the costal border or even to the lower quadrants of the abdomen. It is maximum at onset, constant not colicky, and variously described as cutting, pressing or burning. Morphine is required for its relief. It is intensified by deep breathing, coughing, retching, straining at stool, by bodily motions and extending the thighs. It is lessened by lying on the affected side and flexing the thighs. In a bilateral case the patient may lie on the belly to obtain relief.

As the pain diminishes, in the course of twenty-four hours, it tends to localize to the lumbar region posteriorly. In some cases the pain is referred to the back and flank from the outset. Radiation to the thigh and to the testicle occurred twice.

Tenderness and muscular rigidity are chiefly anterior at first and strongly suggest some acute intraperitoneal disorder. Lumbar percussion, however, causes pain from the beginning. As the acute pain of onset subsides the anterior tenderness and rigidity diminish, costovertebral tenderness and lumbar rigidity become predominant.

According to Schmidt the pain is due to three factors: Increased intracapsular pressure, damage to the renal plexus along the vessels

* Jurgensen's case, Nothnagel's Handbuch, 15, 105, has been excluded because it probably belongs in this category.

and perinephritis. Localization depends on the position of the kidney and the nerve trunks affected. Thus the areas of skin hyperalgesia correspond to the 11th and 12th thoracic and 1st lumbar nerves, and pain may occur in the distribution of the iliohypogastric nerve. Perinephric hemorrhage, edema or thickening are noted in four autopsies.

Associated Vascular Phenomena. In 2 cases there was a history of cerebral embolism producing aphasia in one and hemiparesis in the other, both having completely cleared up. One of my cases had conjunctival petechiæ. Splenic infarcts occurred in 5 cases. Embolism of the femoral, brachial, Sylvian and basilar arteries with their resultant disturbances were also noted in the course of the disease. In the case of chronic endarteritis of the renal vessels (Halperin's case) there was a preceding gangrene of the leg due to disease of the vessels. The case of periarteritis nodosa naturally presented widespread vascular lesions and their effects.

Fever and Leukocytosis. These have been discussed in preceding paragraphs.

Urinary Symptoms and Changes. Bladder function was disturbed in 5 cases; in 1 there was frequency and urgency, in 1 dysuria, in 1 there was great difficulty in voiding, and in 2 there was actual retention. In 3 cases no urinary changes were observed. Marked diminution of urine was noted in 5 cases and complete or almost complete suppression occurred in 2. Albuminuria was present in 9 cases to a marked degree and constituted the most frequent urinary abnormality. Macroscopic hematuria occurred only in the case of periarteritis nodosa, and contrary to the usual belief was not a part of the clinical picture. Erythrocytes were present in 6 cases, and in 6 their absence was specifically recorded. Casts, hyaline, granular or cellular, were present in only 4 cases.

Gastro-intestinal Symptoms are prominent in the clinical picture. Nausea and vomiting at the onset, often persistent and incessant, abdominal distention, constipation or even obstipation, together with the referred pain, tenderness, rigidity of the abdominal muscles, fever and leukocytosis strongly incline one to suspect some acute intraperitoneal disorder requiring immediate surgical intervention. These symptoms are of reflex origin at first and toxic or suburemic afterward.

Diagnosis. To establish a clinical diagnosis of aseptic renal infarction we must find a source for an aseptic embolus (usually valvular disease) or evidence of some constitutional or vascular disease which can produce thrombosis in the renal vessels. In cases of valvular disease we must exclude active bacterial forms of endocarditis. Of the symptomatology emphasis should be placed upon the sudden onset with abdominal pain and gastro-intestinal disturbances. It is characteristic for the pain to be anterior at first and to localize to the kidney region later. Lumbar

percussion, however, elicits pain from the beginning and costo-vertebral tenderness persists after spontaneous pain has ceased. While urinary abnormalities are not constant the presence of albumin and erythrocytes is confirmatory.

Differential Diagnosis. The conditions with which renal infarction may be confused have been indicated in part. The most important is that group of intraperitoneal disorders which requires surgical intervention, namely: Appendicitis, cholecystitis, intestinal obstruction, mesenteric vascular occlusion, perforative peritonitis. A second group of cases is that resulting from obstructive lesions in the ureter occasioned by the presence of or passage of a calculus, blood-clots, inflammatory, leukoplakic or tumor tissue, or by angulation, spasm or inflammatory swelling of the ureter. A third group of cases is that in which renal pain results from sudden increase of intracapsular pressure without obstruction in the urinary passages. Such would be produced by torsion of the vascular pedicle of a floating kidney, compression of the renal veins by retroperitoneal hematomas or inflammatory processes, hemorrhage or congestion in a renal neoplasm. Exacerbations in chronic inflammations of the kidney, as in so-called nephralgie hematurique, produce a similar condition and are specially difficult to differentiate. The renal congestional colics occurring in connection with menstruation as described by French authors also belong in this group.

Schmidt gives a very complete discussion of the details of differentiation.

Prognosis. Of the cases collected, Leube's and my own recovered. The 2 cases of doubtful etiology and pathology recovered after nephrectomy. Bilateral renal infarction occurred in 7 cases, in 5 of which marked suppression or anuria resulted in death. Other embolic lesions appeared to have caused death in 4 cases. In 1 unilateral case the cause of death is not clear; in 1 suppression occurred. Although a patient may recover completely from an attack of renal infarction the underlying disease places him in constant danger of subsequent attacks and of other more serious embolic or thrombotic accidents.

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THE STUDY OF VESTIBULAR NERVE FUNCTION IN MYXEDEMA.*

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The study of the phenomenon of lowered irritability was suggested by the occasional finding of nerve deafness in certain myxedematous patients. As we know, myxedema, or hypothyroidism, is a condition arising from dysfunction of the thyroid gland and manifesting itself by change in weight, cardiac disturbances, dry skin, and other visceral upsets, associated with a certain slowness in response to external stimuli.

From time to time additional data have been added to our knowledge of the interesting condition of myxedema. In recent years light has been thrown on the clinical picture of the condition by the work of Plummer, and Boothby and Sandiford, along the lines of the basal metabolic rate. This work, in conjunction with the isolation of the active principle of the thyroid gland by Kendall, has been of inestimable value in the manipulation of such cases. The literature on the subject has been well combed and brought to date by Lisser, who discusses at length the various changes in the special senses in cretins and in myxedematous patients. St. Lager has reported 5000 cretins in Switzerland and 4000 deaf mutes; 80 per cent of the latter were cretins. Scholz found that 29 per cent of cretins were deaf mutes and that 32 per cent of the others were hard of hearing. Hammerschlag, who was one of the first carefully to investigate the condition, found peripheral ear changes and disturbances in the bone conduction. Various theories and hypotheses have been advanced with regard to the changes taking place, such as ossification of the stapes, impaired development of the epithelial cells in the ductus cochlearis, and so forth. In cases in which the thyroid gland has developed a dysfunction after

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